COMPARISON BETWEEN EFFECTS OF BUPRENORPHINE AND CLONIDINE ADDED TO BUPIVACAINE IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK

A STUDY OF 60 CASES

DISSERTATION SUBMITTED FOR

DOCTOR OF MEDICINE

BRANCH X (ANESTHESIOLOGY)

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY
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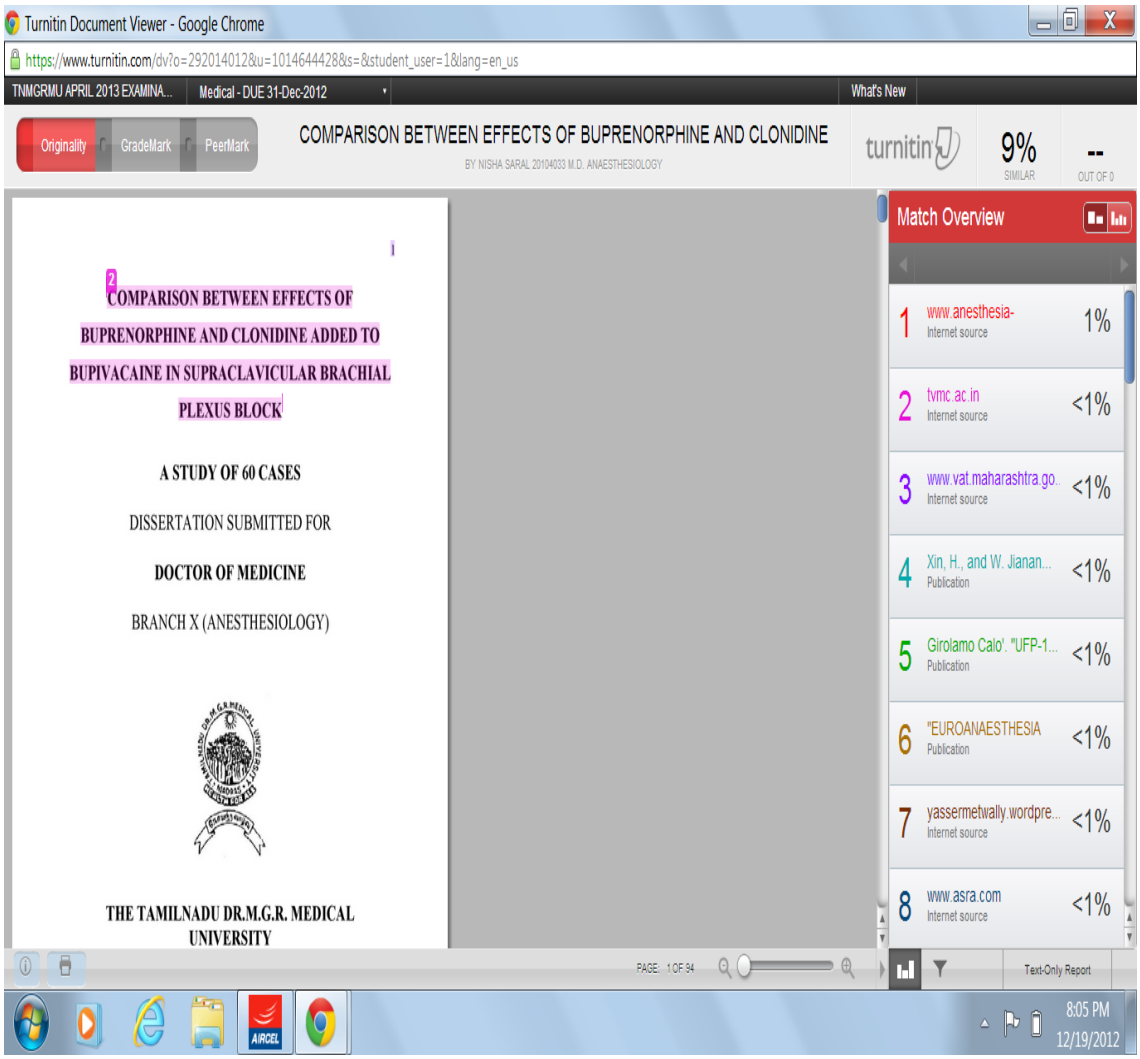
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This is to certify that this dissertation entitled “COMPARISON BETWEEN EFFECTS OF BUPRENORPHINE AND CLONIDINE ADDED TO BUPIVACAINE IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK ” submitted by DR. J.NISHA SARAL to the faculty of ANAESTHESIOLOGY, The TamilNadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement in the award of degree of M.D. Degree, Branch -X (ANAESTHESIOLOGY), for the April 2013 examination is a bonafide research work carried out by her under our direct supervision and guidance.

PROF. DR. A.THAVAMANI, M.D, D.A,

Prof. and Head of the Department,
Department of Anaesthesiology,
Tirunelveli Govt. medical college,
Tirunelveli.

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI,

STATE OF TAMILNADU, INDIA

PIN CODE:627011

Tel: 91-462-2572733, 2572734 Fax: 91-462-2572944

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I, Dr.J.NISHA SARAL, declare that the dissertation titled "COMPARISON BETWEEN EFFECTS OF BUPRENORPHINE AND CLONIDINE ADDED TO BUPIVACAINE IN SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK" has been prepared by me.

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INTRODUCTION:

There are various techniques to anaesthetise a patient. It is really a divine venture to alleviate the pain. Regional techniques are now a days becoming more and more popular because it has its own advantages of providing anaesthesia and pain relief and relaxation required for the surgery pertaining to the area required only without unnecessarily blocking the other sites and avoiding unnecessary complications which can be better avoided just by reducing the poly pharmacy as in general anaesthesia which is not always needed or better to be said it is unnecessary for all the cases.

The generalized thought of general anaesthesia for any surgery has been totally out dated now a days. Thanks to the regional anaesthesia as such because it is feasible many a time and has won very good patient compliance also.

Among the regional techniques we are going to focus mainly on the regional anaesthesia for the upper limb that is the brachial plexus blockade.

There are various available approaches and techniques in brachial plexus blockade. Among them this study concentrates on the supra clavicular technique.

In the supra clavicular technique a huge number of studies have been done at various angles. Many studies have concentrated on the local anaesthetics and its varying concentrations used and their respective effects upon the patients.

ADJUVANTS:

THESE ARE NOTHING BUT THE DRUGS WHICH ARE ADDED TO LOCAL ANAESTHETICS IN ORDER TO

- HASTEN THE ONSET OF BLOCKADE OR
- TO PROLONG THE DURATION OF BLOCK OR
- TO IMPROVE THE QUALITY OF BLOCKADE.

Humpty number of studies have been conducted upon using various and different classes of drugs as adjuvants in brachial plexus block. To enumerate upon the drugs :

- Epinephrine
- Sodium bicarbonate
- Clonidine
- Dexamethasone
- Tramadol
- Dexmedetomidine
- Butorphanol

- Buprenorphine
- Fentanyl
- Alfentanyl

And so on . Each and every drug behaved in a different fashion. [1,2,3,4]

But among all those studies there have not been any study focused on comparison between buprenorphine and clonidine.

Hence this special study is focused to contrast the effects of buprenorphine and clonidine as analgesic adjuvants to bupivacaine in supraclavicular brachial plexus blockade.

AIM & OBJECTIVES

AIM OF THIS PARTICULAR STUDY:

To compare the effectiveness of adding buprenorphine and clonidine as adjuvants to 0.3% bupivacaine in supraclavicular brachial plexus blockade.

OBJECTIVES:

To evaluate

a) The onset time for sensory and motor blockade,
b) The time for complete motor and sensory blockade
c) The total duration of sensory and motor blockade
d) Adverse effects

REALITY BEHIND BRACHIAL PLEXUS: [40]

Brachial plexus - one which supplies nerves to the upper limb. This is formed by the ventral divisions in the 5 TH to the 8 TH Cervical plus the 1 ST thoracic nerves, with its contributions from the 4 th cervical and the 2 nd thoracic roots.

Then roots join to form the

- Superior trunk
- Middle trunk
- Inferior trunk

These trunks now divide into divisions namely

- Anterior division
- Posterior division

The contained three posterior divisions unite to form posterior cord. The major branches of this posterior cord : --- AXILLARY, also the RADIAL NERVE.

The upper two of the anterior divisions -- is derived lateral cord. major branches of the lateral cord are including : MUSCULO CUTANEOUS N., also LATERAL ROOT contribution to MEDIAN NERVE.

Lowest anterior division is forming the medial cord. Medial cord then provides:

MEDIAL ROOT contribution to MEDIAN NERVE, terminating as -- ULNAR NERVE.

sympathetic contributions to this plexus are being derived of so called MIDDLE CERVICAL GANGLION, also STELLATE GANGLION. [5]

VARIOUS TECHNIQUES OF BRACHIAL PLEXUS BLOCK: [6-10]

Four classic routes had been described for blocking the brachial plexus namely:

- Paravertebral – kappis method
- Axillary – Hirschel method
- Infraclavicular – Louis,Bazi method
- Supra clavicular – Kulenkampf method

The first two techniques have been virtually abandoned now days. But the INFRACLAVICULAR AND SUPRACLAVICULAR TECHNIQUES are seen to be achieving a replenishing surge in their use now a days that too with the aid of ultrasound imaging.

The use of the ultrasound imaging is directing the anaesthesiologist towards the targeted nerve providing a live guidance with concurrent visualization of the important vascular plus other vital structures situated there.

This is being used in this era with increasing frequency in aiding the performance of various established regional blocks -- brachial plexus, femoral and sciatic nerves.

The perivascular techniques of brachial plexus block elaborated by WINNIE & COLLINS by axillary, inter scalene or the subclavian approach are found to be the most commonly used techniques. Each is known for its advantages and disadvantages.

When there is an advantage or significant benefit in a Technique, it is a well aware fact also that that kind of advantage may also be overshaded or else set beside that of the disadvantages also which is to be acceptable.

SUPRACLAVICULAR BLOCK:

ADVANTAGES:

- Most compactly arranged form of nerve fibres
- Most intensive blockade achieved
- Smaller volume being required
- Quicker onset achieved
- All of the nerves are being reliably blocked
- Can be performed also if arm is immobilized

DISADVANTAGES:

- Demonstrable PARAESTHESIAS REQUIRED WHICH IS UNPLEASANT FOR THE PATIENT
- 0.5 – 6% of pneumothorax incidence seen
- 40-60% phrenic nerve blockade seen
- 70-90% stellate ganglion blockade recorded
- Possibility of neuritis also seen.

INTERSCALENE BLOCK :

ADVANTAGES:

- This is Ideal for shoulder surgery
- Cervical plexus can also be blocked by this method

- Can be performed even when the arm is immobilized
- Clear landmarks appreciated
- Lower volume sufficient for block
- Lesser chance of pneumothorax than in supraclavicular block

DISADVANTAGES:

- Lower trunk anaesthesia may be missed here
- May also block phrenic nerve, vagus, recurrent laryngeal nerve, also cervical sympathetic nerves.
- Inadvertent entry of drug to other sites and epidural, subdural, spinal anaesthesia have been seen
- Intra vascular injection into vertebral artery may also occur.

AXILLARY BLOCK:

ADVANTAGES:

- Provides Excellent anaesthesia for forearm and hand
- Performance wise Easy technique
- Easily demonstrable landmark
- Safest of all the techniques
- Easiest to do in paediatric group
- Paraesthesia here is not necessary

DISADVANTAGES:

- Insufficient for the shoulder and the upper arm surgery
- To perform this technique , abduction of the arm is required
- Intra vascular injection may be seen
- Haematoma formation

PERIVASCULAR SUBCLAVIAN BLOCKADE:

ADVANTAGES:

- EASY LAND MARKS TO IDENTIFY this.
- Can be done even when the patient's arm is immobilized
- Smaller volume is being needed compared to axillary block
- Lesser chances of leaving the lower trunk unblocked

DISADVANTAGES:

- TO ENSURE THE SUCCESS of this block, paraesthesia needed
- Pneumothorax may be seen
- With large volumes of local anaesthetic this method may also block phrenic nerve, recurrent laryngeal and also cervical sympathetic nerves

Review of Literature

Various Biochemical studies performed to purify the opioid receptor protein but not successful. Since the early ages of 1990s, many molecular biologic studies were elucidating the molecular structures, signal transduction mechanisms in the opioid receptors.

Four very different cDNAs were isolated as the members of this opioid receptor family. Three receptors of them corresponding to the pharmcacologically elaborated μ-, δ-, and κ-opioid receptors. And the fourth receptor was not binding with the opioid ligands with higher affinity. Then, a newer peptide namely nociceptin / orphanin FQ then identified as the endogenous agonist of this fourth member of this opioid receptor family. The μ-receptors are also located in both the brain and the spinal cord and is mediating a various number of pharmacologic actions of opioids.

Further the pharmacological classification of this μ-receptor is as μ_1, μ_2, and $\mu_{3\ which}$ has been proposed. The post-translational modification of this μ-receptor was the molecular basis of the μ-receptor subtypes. **[**11-14]

The Opioids also produce analgesia through a peripheral mechanism. The Immune cells infiltrating the inflammatory site also release endogenous opioid-like substances which act on opioid receptors which is located on the primary sensory neuron. [15, 16, 17, 18, 19, 20]

Buprenorphine is found to produced variable affection of the respiratory adequacy at a dose higher than 3.0 μg/kg to 50% of baseline, in opposition to fentanyl, in which there is dose-dependent respiratory depression and resulting in apnea at doses higher than 2.9 μg/kg. [21]

Sarkar D et al ,[22] compared the effects of adding buprenorphine and fentanyl in supra clavicular block in a controlled study of 75 comparable adults conducted over a period of one year allocated into 3 equal groups with group A as 2 % lignocaine 10 ml with adrenaline + 0.5 5 bupivacaine 20 ml + distilled water 1o ml and group B has added 0.3 mg of Buprenorphine and group C with added 50 mcg fentanyl to the same drug mixture as group A .

He has concluded that the addition of fentanyl had no significant benefits on the duration of analgesia but the addition of buprenorphine has significantly increasing time of devoid of the pain that is analgesia to nearly 1.5 times that of the control group and has observed no significant side effects of adding buprenorphine. [22]

Ashok jadan et al [23] did a study with addition of buprenorphine as adjuvant to 30 ml of 0.3 % bupivacaine and he observed that the addition of 3 mcg / kg of buprenorphine in brachial plexus method of

anaesthesia for the ortho surgery -- the time to obtain complete level of sensory blockade is lengthened but hastens onset to motor block time. It improves the quality in block, lengthens the pain free time that is analgesia but without affecting the duration of motor blockade.

Popping *et al*. [24] in his meta analysis of the randomized trials had showed that there is good pleasing actions in clonidine in the time of pain free period -. They noticed that there is prolongation of motor block which was higher while the same drug then mixed with the bupivacaine as compared to ropivacaine. The least effect is being noted with prilocaine.

There are no published studies which compare the effects of clonidine and buprenorphine as adjuvants in supraclavicular block, that too with 0.3 % bupivacaine. Hence we have chosen this study of comparing the effects of clonidine and buprenorphine as adjuvants in the 30 ml of 0.3% bupivacaine in supraclavicular block.

TECHNIQUES NEEDED TO LOCALISE THE NERVES:

Various tactics are differently said to catch hold of identifying target, that includes "pops," also elicitation of one or more paresthesias, also perivascular or transarterial injection, measured electrical stimulation, and

also field infiltration. Now days, direct imaging by ultrasonography, the fluoroscopy, the computed tomography (CT), and the magnetic resonance imaging (MRI) has also been used. Although there is no definitive study which has identified the best method guiding for needle placement, generalities are possible. The elicitation of a paresthesia is appearing to be equivalent to the electrical stimulation.

The Success rates and the onset times of paresthesia and nerve stimulation techniques are now further improved if the multiple injections are performed. The Transarterial injection is variably successful and a two-injection transarterial technique is also comparable to the single-injection paresthesia or nerve stimulator techniques.

The success rate using a fascial pop or click is variable, it also may be much more reliable in pediatric patients than in the adults. [40]

Now a days the peripheral techniques are being performed under ultrasound guidance moreover with *or* without confirmation of needle placement by the electrical stimulation. Both the approaches involve the visualization of the local anesthetic spread during the injection. [41]

The Ultrasound images of the distribution of the local anesthetic provided the clinicians with the visualization of successful blocks and also have documented why are the single-injection techniques less reliable. [42]

The risk of intravascular injection of large-volume local anesthetic may be reduced by experienced ultra-sonographers' hands.

Supraclavicular Block

CLINICAL USES:

Indications for supraclavicular block are the operations on the elbow, forearm, and hand. The Blockade occurs at the LEVEL OF distal trunk–proximal division level. At this AREA, the brachial plexus is very compact and thus small volume of solution produces a rapid onset of reliable blockade in the brachial plexus. Another additional advantage is that this block can also be performed with the patient's arm in any position.

Ensured supraclavicular blockade requires elicitation of a paresthesia or a motor response. The classic block may be appearing somewhat difficult to describe and to demonstrate. A proposed modification of the technique, the so named plumb-bob approach, decrease the complications and is simplifying the concept of this block.

TECHNIQUE OF BLOCK:

Several anatomic points are important for performance of the supraclavicular approach. The plexus - its three trunks are being clustered vertically overlying the rib number one cephalad and behind the artery of subclavian, that can often be palpated in a slender, relaxed patient.

This neurovascular bundle lying inferior to the clavicle at about the midpoint. The first rib acting as a medial barrier to needle reaching the pleural dome and it is short, broad, and flat, having an anteroposterior orientation at the site of the plexus.

Now ask and instruct the patient to lie in the couch in the normal supine posture and instruct him to look to the opposite side of block intended side by turning his head there. The arm that ought be anesthetized should be adducted, then the hand should be extended toward the ipsilateral knee as far as possible.

In this classic technique, the midpoint of clavicle should be identified and marked first. The posterior border of the sternocleidomastoid may be palpated easily when the patient is asked to raise the head slightly. Then palpating fingers can roll over the belly of the anterior scalene muscle dipping into the interscalene groove, and a mark should be drawn approximately 1.5 to

2.0 cm behind the clavicle ‘s middle point. Palpation of the subclavian artery now at this site confirms the landmark.

Then after appropriate preparation and also development of a skin wheal, now the anesthesiologist stands at the side of the patient facing patient's head. The needle to do block is pricked in this marked site in the downward , inward and behind direction till we are getting the motor response or paresthesia or the rib number one is reached.

While a syringe is attached, this exact orientation causes the needle shaft and syringe to lie almost parallel in a line joining the skin entry site and also the patient's ear. If the first rib is being encountered without elicitation of a paresthesia, the needle then can be systematically walked anteriorly and posteriorly over the rib until the plexus or the subclavian artery is located .The Location of the artery provides a useful landmark and then the needle can be withdrawn and reinserted now in a more posterolateral direction, that generally results in a paresthesia or motor response. After localization of the brachial plexus, aspiration for blood should be done before incremental injections of a total volume local anaesthetic solution of 20 to 30 ml.

The rib is usually contacted at a needle depth of 3 to 4 cm and in an obese patient or in the presence of tissue distortion due to hematoma or injection of

solution, exact depth may exceed the length of the needle. But, before the needle is advanced farther, fine probing in the anterior and posterior directions must be done at the 2- to 3-cm depth if at all paresthesias are not obtained. Multiple injections will improve the quality or shorten the onset of blockade.

The modified plumb-bob approach uses same arrangement and patient positioning, though the needle entry is at the point where the lateral margin of neck muscle sterno cleido mastoid is inserting into clavicle.

After aseptic preparation and raising of a skin wheal, a 22-gauge, 4-cm needle now is inserted while mimicking a plumb-bob as if suspended over the needle entry site. Usually, a paresthesia or motor response is elicited before contacting WITH THE NEEDLE the first rib or artery.

If no paresthesia or motor response is being elicited, the needle is reinserted when angling the tip of the needle upward towards head and then caudad in little steps until the first rib is contacted.

COMPLICATIONS :

Although the block is more difficult to perform in obese patients, there did not appear to be an increased number of complications. The prevalence of pneumothorax after performing a supraclavicular block is about 0.5% to 6% and this diminishes with experience.

The onset of symptoms is sometimes delayed and may take even up to 24 hours. Routine chest x ray after the block is not justified. The supraclavicular technique is best avoided when the patient is not cooperative or he cannot tolerate any degree of respiratory compromise due to the underlying diseases.

Other noted complications include frequent phrenic nerve block (40% to 60%), also Horner's syndrome, and neuropathy. The scene of phrenic or cervical sympathetic nerve blockade usually requires only reassurance. And though the nerve damage can occur, it is uncommon and normally self-limited.

NERVE IMAGING STUDY WITH ULTRASOUND: [27-32]

The Fascicles of peripheral nerves can be detected with a high-resolution ultrasound imaging. The fascicular echotexture is often the most distinguishing feature of nerves namely “honeycomb” architecture. More central nerves, such

as the cervical ventral rami, with fewer fascicles, therefore can appear as monofascicular on ultrasound scans.

One of the most powerful techniques to clinch the nerve fascicles is to slide a broad linear transducer on the area of peripheral target N. being transverse cross section view.

Nerves can appear round, oval, or triangular. Although nerve shape can be changing on course, cross-sectional area is same and constant in the absence of major branching. The Peripheral nerves are pathologically enlarged also by entrapment or in certain other neuromuscular disorders such as Charcot-Marie-Tooth disease of type IA. There is also some evidence to suggest that the patients with diabetic neuropathy are also having enlarged peripheral nerves.

It is true that direct nerve imaging has led to a phenomenal good increase in ultrasound-guided regional anesthesia, but still the identification of other nearby structures like the fascia and other connective tissue is critical in this endeavor.

These significant structures permit favorable distribution of local anaesthetic that the nerve contact with the block needle is not mandated. Successful drug injections must always clarify the borders of the nerve .

ULTRASOUND AND ITS ARTIFACTS IN REGIONAL ANAESTHESIA:

There are several common assumptions in the ultrasound imaging. First of all, the velocity of sound is assumed to be around 1540 msec. This estimate was achieved from measurements on soft tissue at physiological body temperature.

When the local heterogeneities exist, then artifactual bending of the block needle can be seen with sonography, the so-called bayonet artifact. The Speed of sound artifacts relate to the time-of-flight considerations and to the refraction at the interface of tissues with the different speeds of sound. [33,34,35]

Second thing, sound waves are assumed to take a straight linear path to and from the tissue. When this does not occur, the reverberation artifacts occur from the multipath echoes.

Then comet tail artifact is a type of reverberation artifact. At the low receiver gain, the comet tail is seen as a typical tapering series of discrete and clear echo bands just deep to a strongly reflecting structure.

Then spacing between the bands represents the distance seen between the anterior and posterior side walls of the object. Internal clear reverberations which arising from within the object cause the artifact of

comet tail, that is most intensely observed while the object is perpendicular to the beam.

Moreover the pleura is a strong reflector that causes the comet tail artifact. Reverberation echoes are usually seen while strong specular reflections are being received.

During supraclavicular block, the mirror-image artifacts can be observed from the reverberation. While the pleura is adjacent to the subclavian artery, the mirror-image artifacts can occur with gray-scale type sonographic imaging.

Third to say, all reflectors are assumed to be on one central ray of the transducer beam. When this is not occurring true, out-of-plane artifacts are also observed that are slice thickness artifacts.

Definitive proof of the out-of-plane artifacts requires multiple views that are recommended when ambiguities arise.

Not like the adjacent tissue, biologic fluids is not significantly attenuating the sound beam and therefore will cause acoustic enhancement

The Acoustic enhancement artifacts deep to vessels may be erroneously interpreted as the nerves.

For example, acoustic enhancement lying deep to the axillary artery that is in the axilla can mislead .[36-39]

LOCAL ANAESTHETICS: [25]

Local anaesthetics are those drugs which on application topically or as a local injection causing reversible loss of the sensory perception, specially to quote, the pain, in a restricted area where applied of the body.

They block the origination and also conduction of impulses pertaining to nerve mainly at all parts of specific neuron where they have come in the contact without causing any much structural damage to the neuron

Thus to say, not only sensory impulses but motor impulses are also interrupted when this drug is being applied to a mixed nerve, leading on to muscular paralysis and also loss of the autonomic control too.

MAIN DIFFERENCES BETWEEN THE GENERAL ANAESTHESIA AND LOCAL ANAESTHESIA:

- Acting site - CNS for GA,

  Peripheral nerves for LA

- Body area involved - GA – whole body

  LA - applied area

- Consciousness level – lost in GA

  Unaltered in LA

- Care of vital functions – needed in GA

  Not in LA

- Physiological alterations – much in GA

  Less in; LA

- Ill health patient - high risk for GA

  Low risk for LA

- Use in ill cooperating patients – easy for GA, not in LA
- Major surgery - GA –preferred technique

  LA – with some difficulty

The local anaesthetics are actually classified as

- Amide group
- Ester group

The clinically useful type local anesthetic drugs are the weak bases with amphiphilic type of property. A

hydrophilic type of secondary or tertiary type of amine nature on one side and a lipophilic nature of aromatic residue on the other side are joined by an alkyl chain together Through an ester type or amide type linkage.

ESTER LINKED TYPE LOCAL ANAESTHETICS:

- COCAINE
- CHLOROPROCAINE
- PROCAINE
- TETRACAINE
- BENZOCAINE

AMIDE LINKED LOCAL ANAESTHETICS:

- LIGNOCAINE
- BUPIVACAINE
- DIBUCAINE
- PRILOCAINE
- ROPIVACAINE

Pka for the local anaesthetics are:

THE AMIDES :

Bupivacaine AND Ropivacaine	8.1
Lignocaine and Prilocaine	7.8
Etidocaine has	7.7
Mepivacaine has	7.6

ESTERS

Chloroprocaine has	9.0
Procaine has	8.9
Cocaine has	8.7
Tetracaine has	8.2

FEATURES OF AMIDE LOCAL ANAESTHETICS ARE FOUND TO BE IN CONTRAST TO ESTER TYPE LOCAL ANAESTHETICS.

AMIDE LOCAL ANAESTHETICS ARE SPECIFIC IN THIS FOLLOWING SIGNIFICANT APPRECIATING FEATURES:

- Produce much intense and a longer lasting anaesthesia
- Bind to the protein alpha 1 acid glycoprotein in plasma
- Not at all hydrolysed by the enzyme plasma esterases
- Very rarely may cause hypersensitivity reactions, no reported cross sensitivity has been seen with ester type local anaesthetics.

THE MECHANISM OF LOCAL ANAESTHETIC DRUG ACTION:

These drugs are found to block the conduction of nerve impulses by decreasing the penetration of sodium ions in the period of upstroke of the action potential.

And going to the increased concentration level the rise rate of action potential and the top most occurring depolarization reduces leading on to the slowness of conduction.

Thus to say here is THE FAILING OF LOCAL DEPOLARISATION to reach to threshold potential leading to the ensuing conduction interruption.

The predominant active species which is the cationic form of the drug that is able to go near the receptor only if the channel remains in the open conformation at the inner aspect side, and too it binds at significant avid nature to the inactive conformation of the channel.

Hence to say here is, a resting conformation nerve is actually resistant to the action of blocking and the block action is developing rapidly in repeatedly being stimulated nerve.

The frequency of this multiple stimulation is also a tribute factor on which the blockade degree is relying. So the degree of blockade relies mainly on many vital factors.

Greater level of blockade quality has been seen with higher stimulation frequency. And moreover the exposure to a huge concentration of calcium is actually seen to decrease the inactivation of sodium channels and thereby it is lessening the degree of the block. This process of interruption of impulses travel by these local anaesthetic drugs are found to be not mainly due to the hyperpolarisation BUT INDEED THE RESTING POTENTIAL OF THE MEMBRANE IS NOT CHANGED because the channels of potassium will be getting blocked only at a very high local anaesthetic level.

The initial time to start block is linked to the drug's own status

of p -ka. Those drugs with lesser p -ka, to say like

- Lignocaine
- Mepivacaine

are found to be quicker initiation acting, the reason for it is near to thirty to forty percent of that drug is in the base form without dissociation at the normal body level p-H of 7.4 and this exact form is going to penetrate the axon.

The drugs which are actually found to be having high status of p-ka namely:

- Procaine
- Tetracaine
- Bupivacaine

are really having slower initiation of action and its good reason is the lesser percent of only fifteen is in base form without ionization.

The one drug which is really not falling in this trap categorical scene is chloroprocaine which has quicker initiation action even though it is possessing a high rate of p-ka of as high as 9.1.

LOCAL ACTIONS OF THE DRUG:

These drugs clinically have no or to say minimal irritant local action. They decrease the release of acetyl choline also from the endings of motor nerve.

When it is being injected around the motor or mixed nerve it will produce anaesthesia of skin and also paralysis of the voluntary muscular structure supplied by that nerve. The sensory and motor fibres are equally having sensitivity profile.

The sensitivity is determined by the fibre's diameter as well so as what variety of fibre it is. usually the fibres that are thinner are much sensitive than which are in larger compared size. Another thing to notice is that the fibres that are covered by myelin or sheathed are having lesser than expected propensity to get blocked than when compared in the unsheathed fibres.

Fibres also vary by the significant length of axon which must be getting revealed to this drug to deliver an effective interruption of impulses in the travelling propagation.

The fibres which are small are also having a short significant length of axons because in these fibres the alterations in the voltage are travelling passively for compared short distances.

Another thing is more thin axons are found to be having shorter distances of inter nodes and the drugs of local anaesthetic variety enter through the nodes of ranvier only in to the axons. The sodium channel density is very higher in these specific sites, the so called ranvier nodes.

It has been noted already that the block degree also significantly rely on the frequency stimulation. That fact applies well here that is SENSORY FIBRES THAT TOO THE SMALLER TYPES ARE VERY VULNERABLE as they are generating frequency of higher level with longer sustaining action potentials when contrasted with the motor fibres.

When contrasted with the somatic fibres, the autonomic type fibres are much susceptible. While being applied to the tongue, the taste sense of bitter is found to be lost first, next being followed there by the sweetness taste and sour one and the last to all is the salty.

SYSTEMIC ACTION PERTAINED TO THIS DRUG:

ANY OF LOCAL ANAESTHETIC DRUG when it is injected or if it is locally applied, anyway ultimately it is being absorbed entering circulation and naturally producing the effects on the vital systems which also depend on the attained concentration.

The various actions on systems are focused as:

- Neural system
- Cardiac system
- Vascular territorial system

NEURAL SYSTEM:

All these local acting drugs are producing continuous events of stimulation that is sequently followed by depression. The powerful STIMULATING drug on CNS, the so called cocaine drug - in sequence it is causing.

- Euphoria
- Excitedness
- Confusion of mentation
- Restless nature
- Tremor
- Muscle targeted twitches
- Convulsive events
- Unconscious state
- Depression of respiratory adequacy
- Finally - death

These all are seen to be happening in a dose level dependent fashion.

Procaine and also other synthetic locally acting drugs are really having much lesser potency as far as this regard. At the safe doses of clinical usage, they are seen to produce vague apparent CNS effects.

But on the counter level scale to be noticeable thing is that at a higher level doses or while it has accidentally reached in to the vascular space, it is found to produce stimulation of the neural system followed by the phenomenon - depression. On to move to other drugs that is to say on lignocaine which causes drowsy state, lethargy state -- higher doses seen to be producing stimulation of neural system--- then depression.

CARDIAC SYSTEM ACTION:

These are actually the depressing type drugs on the cardiac state. But to say is that conventionally administered dose levels - no notable effects seen. Still at high dose levels, or probable reach inside vascular Channel, then in cardiac tissue:

- Automaticity depressed
- Excitability reduced
- Contractility blunted
- Conductivity slowed
- Effective period of refractoriness lengthened

In this aspect bupivacaine is worse in cardiotoxic profile and it is notorious for:

- Ventricular level tachycardia
- Fibrillation of the ventricles

VASCULAR SYSTEM ACTION OF THE DRUG:

They are in a profile to produce the decline in the blood pressure which is mainly because of the block of the sympathetic tract but at the high level dosages, they are seen to be causing direct stimulated relaxation of the smooth muscular component of the arteriolar area. But it is to be remembering that at doses of toxic reaching levels , it will cause the collapse in the cardio vascular profile .

Procaine and also the relating drugs seen to possess:

- Weaker anti cholinergic
- Also antihistaminic
- Blocking action at ganglion
- Blocking act at neuromuscular area
- Relaxing effect on smooth muscle

Though these were of not much clinical significance.

BUPIVACAINE:[25]

This is a potent and also long acting amide linked local anaesthetic drug which can be used for infiltration, nerve block,epidural and spinal anaesthesia of a long duration. A 0.25 to 0.5 % solution of drug injected epidurally resulted adequate analgesia without that much motor blockade. And hence this has become very popular in obstetrics (mother can actively participate in vaginal delivery) and thus used for post surgery relief of pain by using continuous infusions epidurally.

This possesses a high lipid solubility, distributes high in the tissues than in the blood after giving the subarachnoid / epidural injections and thereby these possesses a lesser chance of reaching the foetus (when being used in labour) to produce neonatal depression.

This drug is more liable to prolong the QTc interval and will induce ventricular level tachycardia or cardiac depression as already mentioned. For this reason, this should not be used for intravenous regional analgesia. Bupivacaine is metabolized in the liver. It provides duration of nerve block to 180 – 360 minutes without any added adjuvants.

The R+ enantiomer Levobupivacaine is equally potent to racemic bupivacaine but less cardiotoxic and also less prone to cause seizures after the accidental intravascular entry of the drug.

BUPRENORPHINE DRUG: [25]

This is actually a synthetic type of drug. This is seen to possess the drug thebaine as its congener. This is having a higher scale of lipid – fat – dissolving nature. It is a mu receptor level action drug. It is possessing the analgesic action. It has twenty five times high potency than morphine drug .

It has slower initiating action but the total acting time is longer. After giving one dosage, the time free of pain is seen to sustain for six to eight hours. If it is used again and multiple occasions it will provide the time free of pain to twenty four hours.

The various seen side effects of this drug buprenorphine are mainly:

- Side effect of sedation
- Side effect of nausea
- Side effect of vomiting
- Side effect of miosis
- Subjective illness
- Side effects in cardiac system
- Less marked constipation
- Side effect of postural hypotension
- Depression of adequacy of respiration

This drug is seen to be having tolerance at a degree of lower status. Also physical type of and also the so called psychological type of the status called dependence is also seen with persons using the drug buprenorphine on a chronic scale use.

The syndrome showing the different symptoms because of withdrawing from its use pertaining to buprenorphine drug is somehow mimicking like the drug called morphine but the clarifying, differentiating things from that are:

- This may take certain days to develop itself
- This is milder
- This is sustaining symptom for longer
- Liability of nature towards abuse rated less than morphine

Naloxone is having ability to reverse its effects just partially only because the drug buprenorphine is tightly appearing bound to its receptor .

This is mainly excreted in the same unchanged form in the bile secretion, so it naturally finds its way of excretion through the faecal residue.

DOSAGES:

- Intra muscular level administered as range of from 0.3 to 0.6 mg
- Subcutaneous dose same as the IM delivery
- Slow administering intravenous dose same as above
- Sublingually also dose is as 0.2-0.4 mg

USES IN THE CLINICAL ASPECT:

- CHRONIC LASTING PAIN AS WITH THE ONCOLOGICALLY AFFECTED PATIENTS
- As in premedication
- For providing pain freeness after surgery
- Also in myocardial infarction

CLONIDINE DRUG: [25,26]

This is actually a derivative of the imidazoline nucleus. This drug is related to the drug called naphazoline, still this has complex profile of actions. This is a partial level agonist with a high level affinity and also high level intrinsic activity at the alpha two receptors, that too in a special subtype called as the alpha two A type of receptors in the stem

of brain. This produces various and major range of effects on haemodynamics found to be resulting from the stimulation of the alpha two A receptors seen mainly post junctionally in the vasomotor site centre in the medulla. This leads to the fall in the outflow of sympathetic throws and thereby fall in the pressure exerted on the vessel lateral walls.

This may lead to bradycardia also because of the raise in the tone of the vagal territory , thus the plasma holding level of nor adrenaline also is declining . This has the vital potential to PREVENT the release of NA directly from the peripheral sites still this effect is not so proving in the doses of clinical use .

UNWANTED EFFECTS OR HARM EFFECTS:

These are of disturbing and of harming nature when given for clinical use, to say are:

- Side effect of depressed mentation
- Side effect of so called sedation
- Disturbed nature of sleep
- Dryness of the area of mouth and also nose , eyes
- Constipation due to the anti-secretory nature action on intestines

- Impotence
- Retention of water and salt
- Bradycardia
- Hypotension when erect posture
- If dose missed in 2 days - threatened rise of pressure on lateral wall of vessel
- A syndrome may occur that is more like pheochromocytoma
- Tachycardia if dose missed
- Anxiety if dose missed
- Headache also occurs if dose missed

INTERACTIONS WITH VARIOUS OTHER PHARMACOLOGICAL PREPARATIONS:

This drug may also like other ones are seen to be reacting with other different drugs namely:

- Tricyclic anti-depressant drug
- Chlorpromazine

Because of these specific interactions, the intended anti-hypertensive nature of the drug may be very much reduced and affected.

USES CLINICALLY:

- In the treatment of moderate level hypertension especially in a combined dose with diuretic.
- In order to treat the syndrome associated with the effect of withdrawing from using opioids.
- Also having the analgesic activity which is made use of to remove the pain of surgery.
- Also as a premedication
- To treat the symptoms of vasomotor effect of menopausal syndrome.
- To treat the loose motions of diabetic neuropathy

MATERIALS AND THE METHODS

STUDY DESIGN:

Prospective randomized controlled observer blinded study.

POPULATION:

60 patients

INCLUSION CRITERIA:

- ASA I, II
- age 20 to 50
- unilateral upper limb orthopaedic surgeries
- both sexes

BLINDING:

It is mainly an observer blinded study.

SAMPLE SIZE-

GROUP ALLOCATION :

- **Group A** (n = 20) – patients receiving 30 ml of 0.3% bupivacaine with 1 ml of normal saline in supraclavicular block as **CONTROL GROUP**
- **Group B** (n = 20) – patients receiving 30 ml of 0.3% bupivacaine with 300 mcg buprenorphine [1 ml] in supraclavicular block as **BUPRENORPHINE GROUP**

- **Group C** (n= 20) - patients receiving 30 ml of 0.3% bupivacaine with 150 mcg clonidine [1 ml] in supraclavicular block as **CLONIDINE GROUP**

EXCLUSION CRITERIA:

- Suspected coagulopathy
- Infection at the site of block
- Age less than 20 and more than 50
- Coexisting diseases like hypertension, diabetes, seizure disorder, liver disease, renal disease, CAHD
- Patient refusal
- Allergy to local anaesthetics and study drugs
- Pregnant women
- ASA III , IV
- Bilateral upper limb procedures
- Pneumothorax even on any one side
- Pulmonary pathology

PRE OPERATIVE EVALUATION:

In all the patients,

- age,
- I.P. No.,
- body weight, and
- baseline vital parameters

were recorded.

History regarding

- previous anaesthesia, surgery,
- any significant medical illness,
- medications and
- allergy were recorded.

Complete physical examination and airway assessment were done. Following laboratory investigations were done: like

- haemoglobin %,
- blood sugar & urea,
- serum creatinine and
- urine analysis.

RANDOMISATION:

The population is allocated into these defined groups according to computer generated random numbers.

STUDY METHOD:

After getting institutional level ethical authorized committee level permission, written level informed, explained consent from the patients, they have been allocated randomly into 3 as:

- Group A taken as control,
- B as Buprenorphine group and
- Group C as Clonidine group as mentioned earlier.

All the adults were given Inj.Atropine 0.02mg per kilo weight i.m. 45 min prior to anaesthesia. Standard monitoring was used during anaesthesia and surgery. HR, MAP and SpO_2 were recorded before surgery and at regular intervals during and after the surgery.

TECHNIQUE:

After sterile preparation of the neck region, 22 G hypodermic needle of 4 cm was used for supraclavicular block by paraesthesia elicitation technique as mentioned in detail in the earlier section. The SOLUTION

was DELIVERED THROUGH THIS NEEDLE checking for repeated careful aspiration for blood if at all to prevent intravascular flow of the drug. While giving the drug was finished, the sensory block was assessed by sensations to pinprick and to evaluate the motor blockade, movements in thumb:

- adduction for ulnar N,
- abduction for radial N,
- opposition for median N,
- flexion of elbow, the supination and the pronation of forearm for musculocutaneous N were assessed.

Hollmen scale made use of to examine sensory as well as motor block. Examination was conducted every one minute after giving the drug and the time taken for initiation of the motor and sensory block was noted. Time for onset is explained as level of at the least grade 2 in measuring hollmen's scale.

Time for complete block is defined as while motor, sensory scores amount to grade 3 according to hollmen scale. Total time of motor block is explained to be the time from the injection of the drug and recovery of muscle power.

Total duration of sensory block is defined -- the time from giving the drug and the tme when complaint pain in the period post-surgery.

When intubation and GA needed for unsuccess of block or not an adequate block that was excluded from the study.

Moreover the intraoperative and post-operative complications were noted which is also to be adequately seen and scored for the necessity of grading the drugs.

HOLLMEN SCALE:

FOR MOTOR BLOCK:

Normal level of function of muscle = 1

Mild level function weakness = 2

Moderate level function weakness =3

total action of muscle lost = 4

Sensory block assessment:

Good level pinprick sensation = 1

perceived -- pointed sharp weak sensation than other hand = 2

Perceived sense- blunt touch with needle=3

Nil sensation to pinpriock =4

STATISTICAL ANALYSIS:

The Randomised three groups were matched by their demographic factors like

- age
- weight

baseline Physiological factors –

- pulse rate,
- MAP and
- SPO_2

By the technique of ANOVA (Analysis of Variance). The differences between them were interpreted by the Post hoc test of Bonferroni. Similarly, the time for sensory block, time for motor block analysed in the anova technique.

Intra and post-operative pulse rate, MAP and SPO_2 at different intervals BEING CLEARLY ANALYSED in the anova technique and interpreted difference by Post hoc test of Bonferroni. The Type of Surgery was analyzed and interpreted by χ^2 test (Chi- square).

The above statistical procedures were performed by the statistical package IBM SPSS statistics- 20. The P – values less than 0.05 ($P<0.05$) were treated as significant in two tail condition.

RESULTS:

group matching:

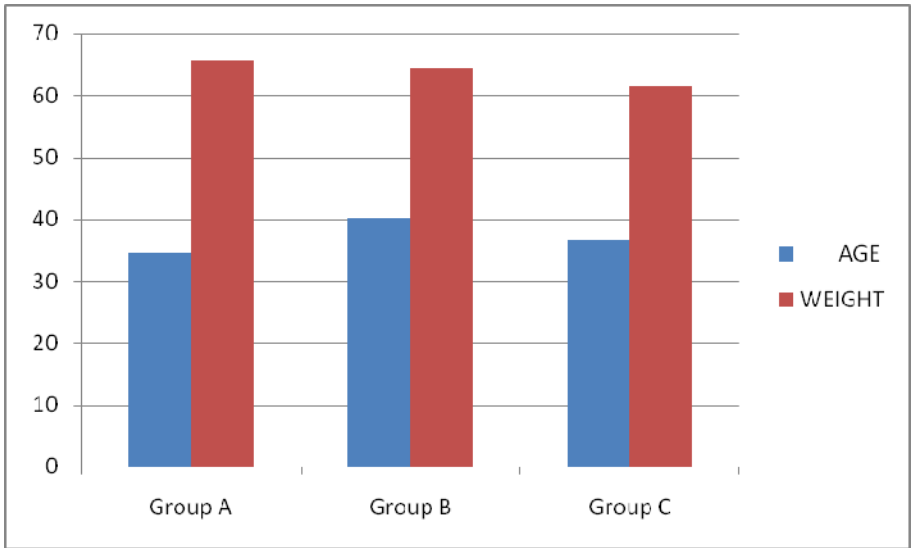
The three groups namely A, B and C were selected with each group encorporating 20 subjects. For randomization the three groups were matched according to their selected and related demographic characteristics such as age and weight.

The baseline Physiological characteristics such as pre pulse rate, MAP and SPO_2 and the type of surgery was also matched. There were no much marked differences among these haemodynamic variables prior to the attempted procedure.

Table -1. Matching of three groups according to their demographic characteristics

	Group	n	Mean	S D	ANOVA 'F'	df	Significance
AGE	A	20	34.5	7.4	2.461	2, 57	P>0.05
	B	20	40.1	8.7			
	C	20	36.6	8.1			
WEIGHT	A	20	65.6	4.3	2.994	2, 57.	P>0.05
	B	20	64.3	5.4			
	C	20	61.4	6.9			

Figure - 1. Comparison of age and weight between the three groups

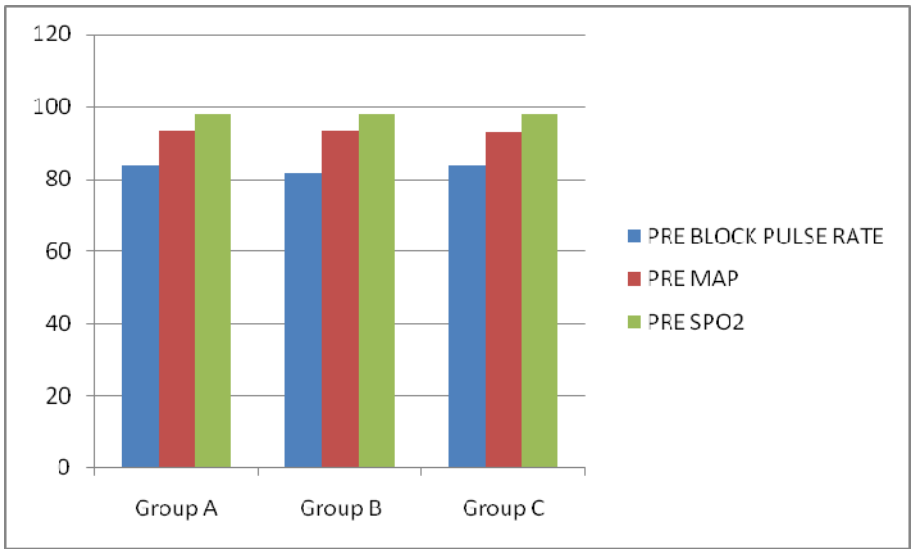


The demographic variables of three groups were matched in the above table -1. The age and weight of three analysed - statistically significant difference --nil (P>0.05).

Table number 2. Matching of three groups according to their physiological characteristics

Variables	Group	n	Mean	S D	ANOVA 'F'	df	Significance
Pre pulse rate	A	20	83.6	4.6	1.136	2, 57	P>0.05
	B	20	81.6	5.5			
	C	20	83.4	3.8			
Pre MAP	A	20	93.4	1.6	0.325	2, 57.	P>0.05
	B	20	93.1	2.5			
	C	20	92.9	1.7			
Pre SPO2	A	20	97.8	0.6	1.715	2, 57	P>0.05
	B	20	98.0	0.7			
	C	20	98.1	0.4			

Figure - 2. Comparison of baseline haemodynamic variables.



Haemodynamic variables of three groups before surgery were matched in the table-2.

In this systematic process of the matching analysis,the

- Pulse rate,
- MAP and
- spo2 were not significantly differed between three groups

(P>0.05).

Table - 3. Matching of the type of Surgery

Type of surgery	A	B	C	Total	χ^2	df	Sig
Bone grafting, distal locking	0	0	1	1			
Buttress plating with k wire fixation	1	0	0	1			
Deroofing & curettage	1	0	0	1			
Implant exit, lrs fixation	0	0	1	1			
Implant exit & im nailing	0	0	1	1	40.871	34	.194
K wire fixation	1	0	0	1			
Oblique osteotomy & k wire fixation	1	0	0	1			
ORIF with berbert screw	0	1	0	1			
ORIF with DCP	1	1	3	5			

ORIF with kwire fixation	1	0	0	1			
ORIF with nailing	0	0	1	1			
ORIF with narrow DCP	2	1	2	5			
ORIF with plate osteosynthesis	7	2	1	10			
ORIF with plate osteotomy	0	1	0	1			
Radial head excision	0	1	0	1			
Refixation of k wire	0	0	1	1			
Total Number of cases	20	20	20	60			

The type of surgery was compared between the groups in table-3. There was no significant association between types of surgery with the groups.

Comparison of PR, MAP, & SPO2 at different time intervals between groups:

Table - 4 Comparison of pulse rate between groups at different time interval.

Pulse Rate at	Group	n	Mean	S D	F	df	Sig (P)
5 Minutes	A	20	84.4	5.3	1.346	2, 57	.248
	B	20	82.8	4.4			
	C	20	85.2	3.6			
10 Minutes	A	20	84.4	6.0	.494	2, 57	.613
	B	20	83.4	4.6			
	C	20	84.8	3.9			
20 Minutes	A	20	84.8	4.1	.974	2, 57	.384
	B	20	83.6	4.4			
	C	20	85.3	3.6			
30 Minutes	A	20	85.1	3.4	1.236	2, 57	.298
	B	20	83.8	4.2			
	C	20	85.5	3.2			

1 Hour	A	20	84.2	4.9	1.576	2, 57	.216
	B	20	83.5	3.8			
	C	20	85.6	3.0			
2 Hour	A	20	84.5	4.6	2.792	2, 57	.070
	B	20	83.2	4.3			
	C	20	86.2	3.0			
3 Hour	A	20	84.9	3.1	1.391	2, 57	.257
	B	20	84.0	4.4			
	C	20	85.9	3.5			
4 Hour	A	20	85.7	2.6	1.634	2, 57	.204
	B	20	84.1	4.3			
	C	20	86.0	3.6			
5 Hour	A	20	85.5	3.7	1.892	2, 57	.160
	B	20	83.2	5.0			
	C	20	85.4	3.5			
6 Hour	A	20	86.0	3.4	.926	2, 57	.402
	B	20	84.4	4.6			
	C	20	85.6	3.8			
8 Hour	A	20	86.0	4.4	.953	2, 57	.392
	B	20	84.2	4.3			
	C	20	85.4	3.8			

12 Hour	A	20	85.5	4.5	1.243	2, 57	.296
	B	20	83.8	4.1			
	C	20	85.7	3.6			
16 Hour	A	20	85.9	4.9	.731	2, 57	.486
	B	20	84.3	3.9			
	C	20	85.2	4.0			
24 Hour	A	20	85.5	4.5	.658	2, 57	.522
	B	20	84.4	3.8			
	C	20	85.8	3.9			

The above table-4 shows the pulse rate between three groups at different time interval. The pulse rates at different interval not significantly altering among these three (P>0.05).

Table - 5. Comparison of MAP between groups at different time interval.

MAP at	Group	n	Mean	S D	F	df	Sig (P)
5 Minutes	A	20	93.6	1.8	1.539	2, 57	.223
	B	20	92.5	2.3			
	C	20	92.6	2.2			

10 Minutes	A	20	93.4	4.0	.103	2, 57	.902
	B	20	93.4	1.6			
	C	20	93.1	1.4			
20 Minutes	A	20	92.6	4.4	.582	2, 57	.562
	B	20	93.4	1.8			
	C	20	93.6	1.9			
30 Minutes	A	20	93.8	0.9	.487	2, 57	.617
	B	20	93.4	1.7			
	C	20	93.8	2.2			
1 Hour	A	20	93.9	1.2	.885	2, 57	.419
	B	20	93.2	1.8			
	C	20	93.5	1.6			
Second hour	A	20	93.9	1.6	.673	2, 57	.514
	B	20	93.2	1.9			
	C	20	93.6	1.9			
Third hour	A	20	93.8	1.6	.143	2, 57	.867
	B	20	93.5	2.9			
	C	20	93.8	1.9			
Fourth hour	A	20	93.4	1.6	1.884	2, 57	.161
	B	20	94.0	1.6			
	C	20	94.3	1.5			

Fifth hour	A	20	93.2	2.1	.630	2, 57	.536
	B	20	93.5	2.2			
	C	20	93.9	1.6			
6thhour	A	20	93.1	2.6	1.764	2, 57	.181
	B	20	93.4	1.9			
	C	20	94.3	1.7			
Eighth hour	A	20	92.9	2.3	1.234	2, 57	.299
	B	20	93.2	1.7			
	C	20	93.9	2.2			
12 Hour	A	20	93.4	2.6	1.007	2, 57	.372
	B	20	92.8	1.8			
	C	20	93.7	1.6			
16 Hour	A	20	93.4	2.9	.044	2, 57	.957
	B	20	93.6	1.6			
	C	20	93.6	1.7			
24 Hour	A	20	93.4	2.6	.622	2, 57	.540
	B	20	93.8	1.6			
	C	20	93.1	16			

The above table-5 states the MAP at different intervals between the three groups. The MAP of three groups were not significantly differing at all intervals between them (P>0.05).

Table - 6. Comparison of spo2 between groups at different time interval.

SPO2 at	Group	n	Mean	S D	F	df	Sig (P)
5 Minutes	A	20	97.4	0.7	.763	2, 57	.471
	B	20	97.6	0.5			
	C	20	97.6	0.7			
10 Minutes	A	20	97.4	0.7	.808	2, 57	.451
	B	20	97.5	0.6			
	C	20	97.6	0.6			
20 Minutes	A	20	97.2	0.7	2.323	2, 57	.107
	B	20	97.5	0.6			
	C	20	97.7	0.6			
30 Minutes	A	20	97.6	0.6	.676	2, 57	.513
	B	20	97.6	0.7			
	C	20	97.8	0.7			

1 Hour	A	20	97.5	0.5	.379	2, 57	.686
	B	20	97.5	0.7			
	C	20	97.6	0.7			
2 Hour	A	20	97.5	0.5	1.267	2, 57	.290
	B	20	97.6	0.7			
	C	20	97.8	0.6			
3 Hour	A	20	97.4	0.5	2.192	2, 57	.121
	B	20	97.8	0.6			
	C	20	97.8	0.8			
4 Hour	A	20	97.4	0.5	2.369	2, 57	.103
	B	20	97.6	0.6			
	C	20	97.7	0.7			
5 Hour	A	20	97.2	0.4	2.019	2, 57	.142
	B	20	97.5	0.5			
	C	20	97.4	0.7			
6 Hour	A	20	97.3	0.6	1.054	2, 57	.355
	B	20	97.5	0.5			
	C	20	97.6	0.9			
8 Hour	A	20	97.5	0.5	1.144	2, 57	.326
	B	20	97.8	0.7			
	C	20	97.7	0.7			

12 Hour	A	20	97.5	0.7	2.019	2, 57	.142
	B	20	97.7	0.6			
	C	20	98.0	0.8			
16 Hour	A	20	97.4	0.8	1.421	2, 57	.250
	B	20	97.7	0.5			
	C	20	97.7	0.6			
24 Hour	A	20	97.5	0.6	.868	2, 57	.425
	B	20	97.8	0.6			
	C	20	97.8	0.7			

The SPO2 of three groups at different intervals was shown in the above table-6. The observed differences between the three groups at different intervals were not found to be statistically significant (P>0.05).

Comparison of Motor Blocks and Sensory blocks:

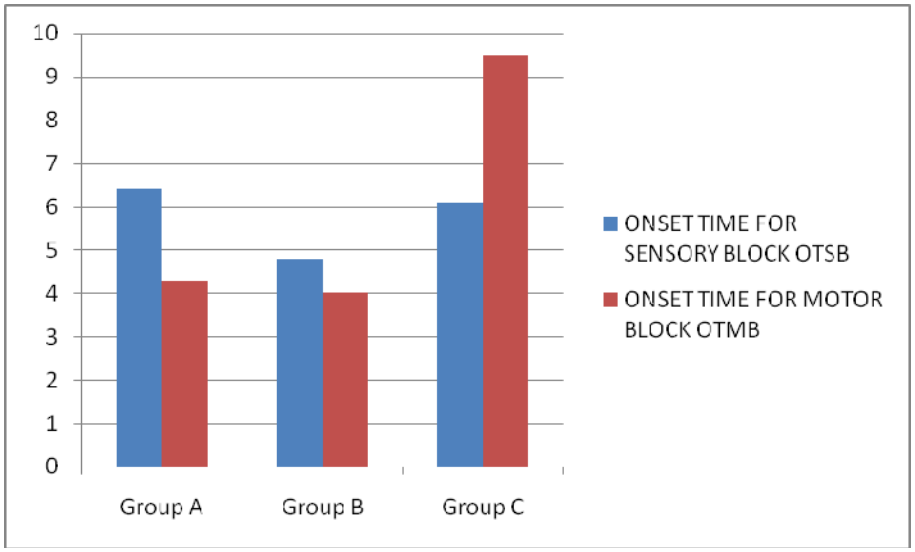
The onset, completion, total sensory, motor block time are analysed between three. The initiation times for sensory, motor blocks of these different sets of population were recorded after the procedure and statistically been analysed between these groups**.**

Table - 7. Comparison of initiation times for sensory, motor blocks between groups.

Blocks	Groups	n	Mean	SD	ANOVA 'F'	df	Sig (P)	Significantly differed groups
OTSB	A	20	6.4	0.5	81.419	2, 57	.000	B differed with A&C. A&C not differed.
	B	20	4.8	0.4				
	C	20	6.1	0.3				
OTMB	A	20	4.3	0.5	609.548	2, 57	.000	A&B not differed. C differed with A&B
	B	20	4.0	0.6				
	C	20	9.5	0.6				

Figure - 3. Comparison of onset time for sensory & motor block.

Onset time in min



The onset time of Sensory block and Motor blocks were shown in the above table -7. The group B significantly differed with A&C in respect of their sensory block on set time and it was comparatively quicker ($P<0.001$).

But, the onset time difference was not significant between the groups A&C ($P>0.05$).

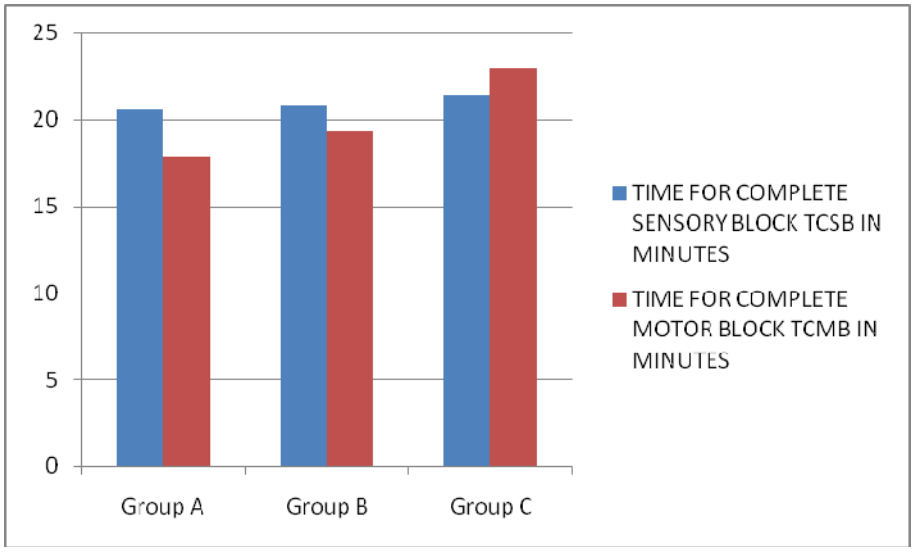
Regarding the onset time of motor block, the difference between A&B groups was not significant (P>0.05). The C group significantly differed with A&B groups and it is longer (P<0.001).

Table - 8. Comparison of Time for Complete Sensory and Motor blocks between groups.

Blocks	Groups	n	Mean	SD	ANOVA 'F'	df	Sig (P)	Significantly differed groups
TCSB	A	20	20.6	1.9	0.364	2, 57	.697	All groups did not differ significantly
	B	20	20.8	3.0				
	C	20	21.4	3.6				
TCMB	A	20	17.8	1.7	19.866	2, 57	.000	A&B not differed. C differed with A&B
	B	20	19.3	2.2				
	C	20	22.9	3.5				

The above table shows time for complete Sensory block and Motor blocks. In respect of their time for complete sensory block all groups did not differ significantly between them (P>0.05).

Figure - 4. Comparison of TCSB & TCMB.

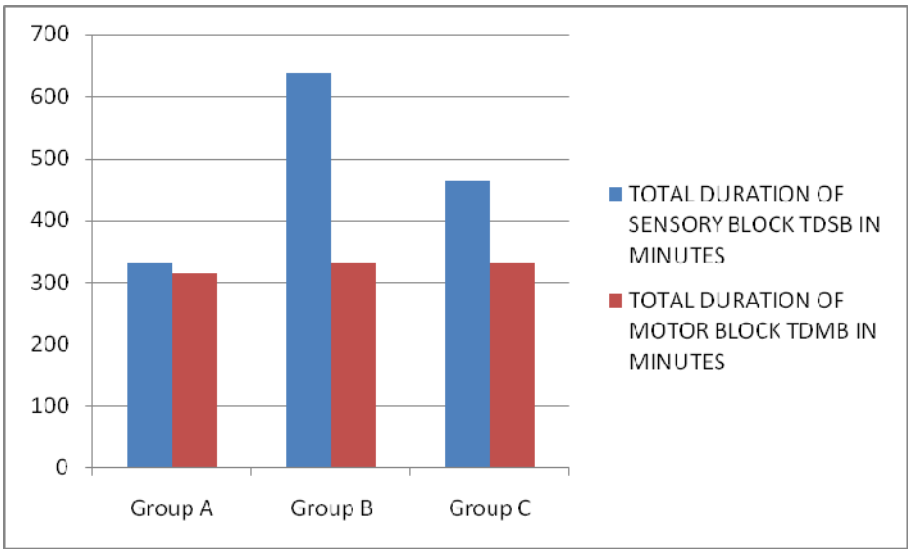


Regarding the time for complete motor block, the difference between A&B groups was not significant (P>0.05). The C group significantly differed with A group (P<0.001) and also differed with B group (P<0.001) and it is comparatively longer.

Table - 9. **Comparison of Total Duration of Sensory and Motor blocks between the groups.**

Blocks	Groups	n	Mean	SD	ANOVA 'F'	df	Sig (P)	Significantly differed groups
TDSB	A	20	330.8	14.5	208.49	2, 57	.000	All groups differed significantly
	B	20	638.5	19.4				
	C	20	464.5	79.1				
TDMB	A	20	313.2	11.9	2.019	2, 57	.142	No significant differences between the groups
	B	20	330.2	11.8				
	C	20	331.8	13.9				

Figure - 5. **Comparison of TDMB & TDSB**



Total duration of Sensory and Motor blocks were compared in table -9. In respect of TDSB, the three groups significantly differed between them ($P<0.001$). It is longer for B than A and C.

It is longer for C than A but less than B.

Regarding the TDMB,

no significant differences were observed between the groups (P>0.05)

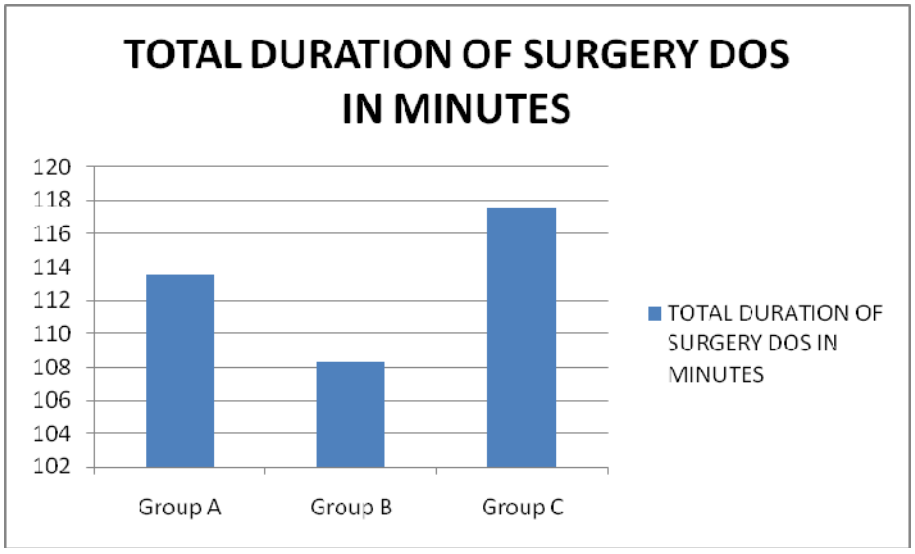
Comparison of duration of surgery:

The durations of surgery between the three groups were compared.

Table - 10. Comparison of surgery duration between the groups.

Groups	n	Mean	SD	ANOVA 'F'	df	Significance
A	20	113.5	16.0	2.029	2, 57	p>0.05
B	20	108.3	13.7			
C	20	117.5	13.9			

Figure - 6. Comparison of DURATION OF SURGERY



The above table-10 compares the duration of surgery between three groups. The durations were not significantly different between the three groups (P>0.05).

DISCUSSION:

In our study 5 cases had failed block and they were eliminated from the study. The mean ages of A, B, and C groups were 34.5±7.4, 40.1± 8.7 and 36.6±8.1 years respectively. They did not differ between them significantly(P>0.05). The mean weights of three groups were 65.6±4.3, 64.3±5.4 and 61.4±6.9 Kgs respectively. The weights of the three groups were not significantly different (P>0.05).

The pre pulse rates of three groups namely A, B, and C were 83.6 ± 4.6, 81.6±5.5 and83.4±3.8 respectively. Even fortunately the sex ratio of each group were also similar making the matching unnecessary. The pre MAP were 93.4 ± 1.6, 93.1±2.5and 92.9±1.7 respectively.

The Pre SPO2 was 97.8±0.6, 98.0±0.7 and 98.1±0.4 respectively. The pre Physiological variables between the three groups were not significantly different. Hence, the three groups were comparable groups.

The intra and post-operative pulse, MAP and SPO_2 of three groups at different time interval like 5 Minutes, 10 Minutes, 20 Minutes, 30 Minutes, 1 Hour, 2 Hours, 3 Hours, 4 Hours, 5 Hours, 6 Hours, 8 Hours, 12 Hours, 16 Hours and 24 Hours were analysed statistically. The pulse, MAP and SPO_2 recorded in the above time were not significantly different ($P>0.05$).

The onset time of sensory blocks of A group was 6.4 ±0.5 minutes, B group was 4.8±0.4 and C group was 6.1±0.3 minutes. The OTSB of B group was significantly lower than A & C groups (4.8±0.4 < 6.4 ±0.5& 6.1±0.3 and $P<0.05$). The onset time of motor blocks of A group was 4.3± 0.5, B group was 4.0 ±0.6 and C group was 9.5±0.6 minutes.

The OTMB of C group was significantly higher than A&B groups (9.5±0.6 > 4.3± 0.5 &4.0 ±0.6 and $P<0.05$).The above findings have differed from the observations of the study conducted by Jadon et al in which the onset time of motor block alone is hastened by adding buprenorphine but the onset time for sensory block was prolonged. [23]

The Time for Complete Sensory Block of A group was 20.6 ±1.9, B group was 20.8±3.0 and C group was 21.4±3.6 minutes. The TCSB of three groups were not significantly different (20.6 ±1.9=20.8±3.0=21.4±3.6 and $P>0.05$). The Time for Complete Motor Block of A group was 17.8 ± 1.7, B group was 19.3± 2.2 and C group was 22.9 ± 3.5 minutes.

The TCMB of C group was significantly higher than A&B groups (22.9 ± 3.5> 17.8 ± 1.7&19.3± 2.2 and P<0.05

The Total Duration of Sensory Block of A group was 330.8 ±14.5, B group was 638.5 ± 19.4 and C group was 464.5± 79.1minutes. The TDSB of B group was significantly longer than the C and C longer than A groups (638.5 ± 19.4> 464.5± 79.1>330.8 ±14.5 and P<0.001).

The Total Duration of Motor Block of A group was 313.2±11.9, B group was 330.2 ± 11.8 and C group was 331.8± 13.9 Minutes and thus did not differ significantly.

The Durations of surgery between the three groups were not significantly different and hence they were equal (113.5±16.0 ≈ 108.3±13.7 ≈ 117.5±13.9 and P>0.05).

Adverse effects were also watched for in all the three groups and no significant, serious effects were found except for nausea in one case in the B group which is not significant.

From the above results and discussions, by considering the non-significant variations of PR, MAP and SPO2, the significant longer duration of TDSB and shorter onset time for motor and sensory block, the group B is considered as better than the other two groups.

Bazin et al, had observed a similar duration of analgesia (median 20 hrs) on buprenorphine administration. In a similar way Candido et al has observed duration of analgesia produced with buprenorphine 3 times longer than that produced by local anaesthetics alone.

Wajima et al has also found satisfactory and prolonged analgesia with butorphanol administered as continuous intra brachial infusion. The prolonged analgesic duration observed with buprenorphine may be attributed to its high affinity for the mu opioid receptor and its high lipid solubility.

This factor favours easy penetration through the axonal myelin and nerve membrane. The other factor that might have influenced the protracted analgesia of buprenorphine, was

- its potency .
- Buprenorphine is 33 to 35 times more potent than morphine.

Side effects:

In our study no major adverse effects have been noted. Though some studies have reported side effects of bradycardia with clonidine, there were no significant changes in the blood pressure or heart rate in the clonidine group as well as in the buprenorphine group. And there were no complaints of nausea and vomiting except one in buprenorphine group.

SUMMARY:

Onset time for sensory block

It is shorter in buprenorphine group than the other groups.

Onset time for motor block

It is similar in the control and buprenorphine group but it is longer in clonidine group than the other groups .

Time for complete sensory block

This came out as similar in all the groups.

Time for complete motor block

It is found to be longer in clonidine group than the other 2 groups which have similar TCMB.

Total duration of motor block -

It is almost similar in all the groups.

Total duration of sensory block

Buprenorphine group has the longest duration of sensory block then comes the clonidine group and then the control group.

CONCLUSION:

We conclude from this randomized controlled study that buprenorphine

- hastens the onset of sensory block,
- significantly prolongs the duration of analgesia compared to clonidine
- without any significant effect on onset of motor block and duration of motor block and
- without any significant adverse effects.

Hence it is clear from this study that **buprenorphine is a better adjuvant than clonidine in supraclavicular brachial plexus block.**

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PROFORMA

Case NO- I.P-no-

NAME AND ADDRESS OF THE PATIENT

Age- : Sex D.O.ADMISSION

History in brief-

Clinical diagnosis/ indication.

Examination in brief-

A. Vitals
a. Pulse b .B.P- C**.AIRWAY-ASSESSMENT**

B. Systemic examination

Baseline haemodynamics :

PR: BP: SPO2:

INVESTIGATIONS
Hb% BT CT Blood urea Serum creatinine
RBS
Urine examination- Albumin Sugar Microscopy

Chest X-ray ECG **ASA** Grading-

Surgical procedure

Informed written consent.

Name of the patient- I.P. no-

I ,---------- in my full sense give my wholehearted consent for the surgery under brachialplexus block with clonidine and buprenorphine for study purpose. I agree that no responsibility will be attached to the surgeon or anaesthetist. I have been explained about the procedure in my own language.

signature of the patient / guardian

PROCEDURE—supraclavicular block

PARAMETERS OBSERVED-

1. onset time for sensory block OTSB
2. onset time for MOTOR block OTMB
3. time for complete sensory block
4. time for complete motor block
5. total duration of sensory block
6. total duration of motor block

Intra op &Postoperative Monitoring:

Time after block	Heart rate	Blood pressure	Spo2

Side Effects

Nausea +/-
Vomiting +/-
Drowsiness +/-
Pruritis +/-

GROUP A

S.NO	AGE	SEX	WT	TYSURG	BASE LINE			PULSE RATE													MAP					
					PR	MAP	SPO2	5M	10M	20M	30M	1H	2H	3H	4H	5H	6H	8H	12H	16H	24H	5M	10M	20M	30M	1H	2H
1	32	F	58	oblique osteotomy & k wire fixation	76	90	99	72	70	76	78	72	73	79	80	76	77	74	73	72	73	90	82	80	93	93	92
2	38	M	65	ORIF with plate osteosynthesis	86	96	98	89	88	86	87	89	88	83	89	89	87	90	83	89	83	96	95	94	94	95	96
3	34	M	70	ORIF with DCP	87	95	98	88	89	88	87	85	86	84	87	87	89	88	87	87	87	95	96	94	95	95	96
4	24	M	68	ORIF	80	96	97	86	82	85	84	86	87	88	87	84	86	88	87	89	87	98	97	96	95	96	96
5	33	M	70	ORIF with asian DCP	86	93	98	86	88	87	86	85	90	89	88	89	87	88	88	86	88	93	94	95	96	93	93
6	21	M	66	ORIF with plate osteosynthesis	79	93	98	79	78	78	80	80	80	81	83	84	84	83	84	85	84	93	94	93	94	95	95
7	33	M	67	ORIF with narrow DCP	80	94	97	80	82	84	86	84	84	84	83	83	88	88	87	86	87	94	95	94	94	94	94
8	50	M	69	ORIF with plate osteosynthesis	80	92	97	84	85	86	84	84	84	85	85	85	86	87	87	87	87	92	91	91	93	93	93
9	28	M	65	ORIF with plate osteosynthesis	89	94	97	88	87	87	87	87	87	88	88	89	89	89	87	88	87	94	95	94	94	94	94
10	43	M	71	k wire fixation	89	93	97	88	89	87	88	89	87	88	88	87	87	88	89	89	89	93	94	95	93	92	92
11	40	M	69	ORIF with plate osteosynthesis	88	94	98	89	88	87	88	86	86	86	86	88	89	87	87	87	87	94	95	94	93	93	92
12	29	M	65	ORIF with asian DCP	86	94	97	87	88	88	89	89	87	88	87	88	87	87	88	89	88	94	95	94	93	93	94
13	33	M	68	ORIF with plate osteosynthesis	80	93	98	86	88	87	86	86	87	86	87	87	86	88	88	89	88	93	94	94	94	95	95
14	37	M	65	deroofing & curettage	86	93	98	84	85	86	84	84	84	85	85	85	86	87	87	87	87	94	95	94	94	94	94
15	25	F	55	ORIF	87	95	98	88	89	88	87	85	86	84	87	87	89	88	87	87	87	95	96	94	95	95	96
16	34	M	60	buttress plating with k wire fixation	79	93	98	79	78	78	80	80	80	81	83	84	84	83	84	85	84	93	94	93	94	95	95
17	39	M	65	ORIF with asian DCP	76	90	99	72	70	76	78	72	73	79	80	76	77	74	73	72	73	90	82	80	93	93	92
18	34	M	66	ORIF with plate osteosynthesis	89	93	97	88	89	87	88	89	87	88	88	87	87	88	89	89	89	93	94	95	93	92	92
19	34	M	70	ORIF with narrow DCP	80	93	98	86	88	87	86	86	87	86	87	87	86	88	88	89	88	93	94	94	94	95	95
20	48	M	60	ORIF with kwire fixation	88	94	98	89	88	87	88	86	86	86	86	88	89	87	87	87	87	94	95	94	93	93	92

GROUP A continued

MAP								SPO2														MIN						
3 H	4 H	5 H	6 H	8 H	12 H	16 H	24 H	5 M	10 M	20 M	30 M	1 H	2 H	3 H	4 H	5 H	6 H	8 H	12 H	16 H	24 H	OTS B	OTM B	TCS B	TCM B	DO S	TDM B	TDS B
91	90	88	86	87	87	86	87	99	96	97	98	97	98	98	98	98	98	98	99	99	99	6	4	18	15	105	330	345
96	94	95	94	95	96	96	96	98	98	99	99	98	98	98	97	97	97	97	98	97	98	7	4.5	23	16	110	300	331
96	96	95	95	95	96	96	96	98	97	97	97	98	97	97	98	97	97	97	98	98	98	7	4	19	20	120	315	340
96	96	96	95	94	95	96	95	97	97	97	97	97	98	97	98	97	98	98	97	97	97	6	4	22	19	90	320	335
93	94	94	94	95	95	93	95	98	98	97	97	97	97	98	97	97	98	98	97	97	97	5.5	4	21	18	120	315	330
95	94	93	93	93	94	95	94	97	98	98	98	98	98	98	97	97	97	97	97	96	97	6	5	20	17	110	310	345
95	93	93	94	94	95	95	95	97	98	97	97	97	97	97	97	97	98	98	98	97	98	6.5	4	18	17	105	330	300
93	93	93	93	93	92	91	92	96	97	97	97	97	97	97	97	98	98	98	98	98	98	6.5	4	22	18	105	330	300
93	93	93	94	94	95	95	95	98	98	98	97	97	97	98	97	97	98	98	98	97	98	6.5	5	21	19	120	315	340
92	92	93	92	92	91	93	91	97	97	97	98	98	98	98	98	97	97	98	97	98	97	6	4	20	15	135	300	330
93	93	93	94	92	94	93	94	97	97	98	98	98	98	97	97	97	96	97	97	97	97	6	4	23	20	135	300	330
94	93	96	95	95	94	94	94	97	98	97	98	98	97	97	97	97	97	97	97	96	97	6.5	5	19	20	120	315	340
94	94	94	95	93	94	94	94	97	98	96	97	97	97	97	97	97	97	97	97	98	97	7	5	22	18	110	300	330
95	93	93	94	94	95	95	95	97	98	97	97	97	97	97	97	97	98	98	98	97	98	6.5	4	18	15	70	330	300
96	96	95	95	95	96	96	96	98	97	97	97	98	97	97	98	97	97	98	98	98	98	7	4	20	16	120	315	340
95	94	93	93	93	94	95	94	97	98	98	98	98	98	98	97	97	97	98	97	96	97	6	5	21	19	110	310	345
91	90	88	86	87	87	86	87	99	96	97	98	97	98	98	98	98	98	98	99	99	99	6	4	23	17	105	330	345
92	92	93	92	92	91	93	91	97	97	97	98	98	98	98	98	97	97	97	97	98	97	6	4	22	20	135	300	330
94	94	94	95	93	94	94	94	97	98	96	97	97	97	97	97	97	97	97	97	98	97	7	5	22	19	110	300	330
93	93	93	94	92	94	93	94	97	97	98	98	98	98	97	97	97	96	97	97	97	97	6	4	18	18	135	300	330

GROUP B

S.NO	AGE	SEX	WT	TYSURG	BASE LINE			PULSE RATE													MAP					
					PR	MAP	SPO2	5M	10M	20M	30M	1H	2H	3H	4H	5H	6H	8H	12H	16H	24H	5M	10M	20M	30M	1H	2H
1	45	M	68	ORIF	75	100	99	79	78	77	76	76	75	76	79	77	80	82	78	79	79	98	97	98	97	95	96
2	31	M	67	ORIF	88	95	98	89	88	87	89	88	88	89	89	89	89	89	89	89	89	95	96	96	96	97	96
3	50	M	66	ORIF with asian DCP	70	93	97	76	75	76	78	78	79	78	76	72	77	76	76	78	79	93	93	94	94	95	96
4	46	M	70	ORIF	86	96	98	87	88	89	88	88	88	87	88	89	86	86	86	86	87	94	94	95	95	96	95
5	22	M	60	ORIF with berbert screw	87	88	98	87	86	86	86	86	87	87	87	88	88	88	87	86	86	97	96	97	97	96	98
6	40	M	70	ORIF with asian DCP	79	93	99	80	82	87	87	88	89	87	86	86	86	86	86	89	89	92	93	93	94	93	93
7	41	M	60	ORIF	80	95	99	84	86	84	85	83	83	89	89	87	87	88	85	84	84	93	94	93	92	92	91
8	50	M	66	ORIF	76	92	97	79	78	80	83	82	81	83	84	79	78	78	78	85	84	90	93	94	94	93	92
9	45	M	50	ORIF with plate osteosynthesis	85	90	97	86	87	85	84	84	84	85	83	82	88	85	86	86	87	90	92	91	93	91	92
10	47	M	60	ORIF	88	93	98	85	87	87	86	85	84	85	85	86	87	87	87	88	85	93	92	92	92	94	93
11	40	F	60	ORIF	86	92	98	87	87	88	88	87	86	86	87	87	88	88	87	86	88	90	91	92	92	93	93
12	38	F	55	ORIF with plate osteosynthesis	77	94	98	76	78	79	78	80	78	78	78	78	79	80	82	79	79	92	94	93	92	91	92
13	21	M	66	ORIF	86	92	98	86	87	87	87	87	89	88	88	88	89	87	87	87	87	92	93	92	93	92	92
14	35	M	68	ORIF with narrow DCP	76	93	98	78	77	76	76	76	76	76	77	78	78	78	78	76	77	93	94	93	92	93	93
15	30	M	68	ORIF with DCP	80	95	99	84	86	84	85	83	83	89	89	87	87	88	85	84	84	93	94	93	92	92	91
16	50	M	66	ORIF	76	92	97	79	78	80	83	82	81	83	84	79	78	78	78	85	84	90	93	94	94	93	92
17	40	M	68	ORIF	85	90	97	86	87	85	84	84	84	85	83	82	88	85	86	86	87	90	92	91	93	91	92
18	50	M	70	ORIF with plate osteotomy	88	93	98	85	87	87	86	85	84	85	85	86	87	87	87	88	85	93	92	92	92	94	93
19	40	M	66	radial head excision	86	92	98	87	87	88	88	87	86	86	87	87	88	88	87	86	88	90	91	92	92	93	93
20	40	M	62	ORIF	77	94	98	76	78	79	78	80	78	78	78	78	79	80	82	79	79	92	94	93	92	91	92

GROUP B continued

MAP								SPO2														MIN						
3 H	4 H	5 H	6 H	8 H	12 H	16 H	24 H	5 M	10 M	20 M	30 M	1 H	2 H	3 H	4 H	5 H	6 H	8 H	12 H	16 H	24 H	OTS B	OTM B	TCS B	TCM B	DO S	TDM B	TDS B
95	96	97	97	98	98	98	97	97	96	97	97	97	97	98	98	98	98	98	97	97	97	4	3.5	17	16	130	330	645
96	96	95	94	95	95	96	96	97	98	98	98	98	98	98	98	98	98	98	98	98	98	4.25	3.75	18	18	110	315	630
96	93	94	94	95	94	95	96	98	98	97	98	98	97	98	99	98	98	98	98	98	98	3.5	2.5	20	20	105	345	660
96	96	96	96	94	95	96	96	98	98	98	98	97	97	97	97	97	98	98	98	99	98	5	4.75	25	22	115	330	615
98	94	97	94	93	93	94	95	97	98	98	98	98	98	98	98	98	97	97	97	97	98	4.75	3	26	21	120	345	645
94	95	95	94	93	93	93	93	98	98	99	99	98	98	98	98	97	98	99	98	98	98	4.5	3.5	19	17	120	345	660
94	95	96	96	94	92	93	91	98	98	98	99	99	98	98	98	98	98	99	99	98	98	5	4	22	19	100	315	615
92	92	91	93	92	92	91	93	97	97	97	97	97	97	97	97	97	98	98	97	97	97	4.75	3.75	21	20	100	330	630
92	92	92	92	92	90	93	94	98	97	97	97	97	97	97	97	97	98	98	98	98	98	5	4	23	21	90	315	630
95	96	90	90	91	93	93	93	98	97	97	97	97	97	98	98	98	97	97	98	98	97	5.25	4.75	25	22	100	345	670
93	92	93	93	94	92	94	94	97	97	97	97	97	99	98	98	98	97	98	98	98	99	5	4.25	18	17	135	340	615
93	94	94	94	93	92	93	94	98	98	98	98	98	98	99	97	97	97	97	97	97	98	5.25	4.75	17	18	100	330	660
83	93	91	92	91	94	93	93	98	98	97	97	97	97	97	97	97	97	97	97	97	98	4.75	3.75	18	16	90	330	630
94	94	93	92	92	92	94	93	97	98	98	98	97	98	98	98	97	97	98	98	98	97	5	4	25	16	120	315	645
94	95	96	96	94	92	93	91	98	98	98	99	99	98	98	98	98	98	99	99	98	98	5	4	20	22	100	315	615
92	92	91	93	92	92	91	93	98	97	97	97	97	97	97	97	97	98	98	97	97	97	4.75	3.75	22	21	105	330	630
92	92	92	92	92	90	93	94	98	97	97	97	97	97	97	97	97	98	98	98	98	98	5	4	18	17	90	315	630
95	96	90	90	91	93	93	93	98	97	97	97	97	97	98	98	98	97	97	98	98	97	5.25	4.75	18	22	100	345	670
93	92	93	93	94	92	94	94	97	97	97	97	97	99	98	98	98	97	98	98	98	99	5	4.25	20	20	130	340	615
93	94	94	93	93	92	93	94	98	98	98	98	98	98	99	97	97	97	97	97	97	98	5.25	4.75	24	21	105	330	660

GROUP C

S.NO	AGE	SEX	WT	TYSURG	BASE LINE			PULSE RATE													MAP					
					PR	MAP	SPO2	5M	10M	20M	30M	1H	2H	3H	4H	5H	6H	8H	12H	16H	24H	5M	10M	20M	30M	1H	2H
1	25	F	55	ORIF with narrow DCP	86	89	98	86	87	88	87	87	87	87	87	88	88	88	87	88	88	88	90	92	92	91	90
2	23	M	60	bone grafting , distal locking	88	90	98	87	87	87	88	87	87	87	88	87	87	88	89	88	89	90	92	93	91	92	93
3	43	F	50	ORIF with DCP	82	92	98	83	84	82	84	85	87	88	87	85	84	84	82	85	83	92	93	94	90	91	92
4	38	M	60	ORIF with DCP	86	95	98	88	87	89	89	89	89	90	89	89	89	90	89	88	88	95	94	98	99	97	98
5	30	M	70	ORIF with asian DCP	81	97	98	87	86	85	85	84	86	86	87	87	88	89	88	89	89	96	95	95	95	96	96
6	32	M	60	ORIF	88	94	98	88	89	89	89	88	87	87	87	86	86	86	88	87	88	96	95	96	94	94	94
7	45	M	66	ORIF with asian DCP	80	93	98	87	84	85	86	86	87	87	86	85	85	86	86	85	88	94	93	92	96	95	95
8	40	M	68	ORIF	85	94	99	85	87	86	86	87	87	87	89	88	88	84	84	85	85	93	94	94	95	93	92
9	44	M	70	ORIF with asian DCP	79	92	98	79	80	83	82	81	80	79	78	78	78	77	80	76	78	92	93	94	93	93	92
10	45	M	65	ORIF with DCP	86	93	98	88	86	87	87	88	89	87	87	87	89	88	89	88	88	92	93	93	94	94	94
11	38	M	70	ORIF with plate osteosynthesis	88	92	99	87	86	86	87	87	88	88	89	87	87	88	89	89	90	90	93	92	92	93	94
12	50	M	60	ORIF with narrow DCP	76	92	98	78	76	76	78	80	82	78	79	78	79	80	82	83	81	92	91	95	96	94	93
13	49	M	70	implant exit & im nailing	87	93	98	87	88	89	87	88	88	89	89	89	90	89	88	87	86	90	92	94	95	95	95
14	28	M	60	ORIF	78	94	98	78	76	77	78	79	80	82	83	83	80	82	78	78	78	92	91	90	91	92	92
15	40	M	50	ORIF with nailing	86	92	97	88	88	87	87	87	89	90	88	87	87	87	88	87	89	93	94	90	92	91	96
16	35	M	68	ORIF	82	94	98	88	89	89	89	88	87	87	87	86	86	86	88	87	88	96	95	96	94	94	94
17	33	M	60	refixation of k wire	80	93	98	87	84	85	86	86	87	87	86	85	85	86	86	85	88	94	93	92	96	95	95
18	26	M	55	implant exit , lrs fixation	85	94	99	85	87	86	86	87	87	87	89	88	88	84	84	85	85	93	94	94	95	93	92
19	29	M	60	ORIF	79	92	98	79	80	83	82	81	80	79	78	78	78	77	80	76	78	92	93	94	93	93	92
20	38	M	50	ORIF	86	93	98	88	86	87	87	88	89	87	87	87	89	88	89	88	88	92	93	93	94	94	94

GROUP C continued

MAP								SPO2														MIN						
3 H	4 H	5 H	6 H	8 H	12 H	16 H	24 H	5 M	10 M	20 M	30 M	1 H	2 H	3 H	4 H	5 H	6 H	8 H	12 H	16 H	24 H	OTS B	OTM B	TCS B	TCM B	DO S	TDM B	TDS B
90	93	92	93	89	92	91	93	98	97	98	98	97	99	99	98	98	98	98	98	98	99	6	10	15	22	115	330	420
93	92	92	93	93	94	92	92	97	98	98	97	98	98	97	97	97	98	98	98	98	99	6.5	9	25	25	130	330	435
93	94	94	93	94	93	92	93	98	97	97	97	97	98	98	98	98	98	98	98	98	98	6.5	9.5	20	28	135	345	460
97	97	96	93	98	98	97	98	99	98	99	98	98	98	99	99	98	98	98	98	98	98	6	9.25	22	23	90	345	405
95	97	96	93	94	96	95	95	97	98	98	98	98	98	97	97	98	98	98	99	99	99	5.5	10	25	24	110	315	510
95	96	95	93	96	94	96	95	97	97	98	98	97	98	98	98	98	99	99	99	98	98	6.25	9.5	26	26	130	330	450
95	95	94	93	93	93	92	92	98	98	98	99	98	98	97	98	97	97	97	97	97	97	6.5	9	27	27	125	315	645
94	95	95	93	96	94	93	92	97	98	98	98	97	97	99	97	97	97	97	98	98	99	6	9.5	16	28	125	345	435
91	94	92	93	91	91	93	92	97	97	97	97	98	98	98	99	96	96	97	97	98	98	6.25	9.75	19	25	120	315	440
93	94	95	93	95	95	95	94	98	99	97	97	97	97	98	98	98	98	98	99	97	97	6.5	8.5	25	23	90	330	405
93	92	91	93	92	93	95	93	97	98	98	98	98	98	99	97	97	97	97	97	97	97	5.75	9.5	20	22	110	315	450
93	93	96	93	97	95	93	92	98	97	97	97	98	98	97	99	99	99	99	98	98	98	6	9.75	22	25	135	350	600
96	93	92	93	93	94	93	92	99	98	98	98	97	97	98	98	97	97	97	97	97	97	5.75	8.75	16	15	125	330	500
93	93	93	93	93	93	93	93	97	97	97	99	99	99	97	97	97	97	98	99	99	98	6	11	18	18	130	360	400
97	94	95	93	93	92	92	92	97	98	98	98	97	97	97	97	97	98	98	98	98	97	5.75	10	19	20	120	345	360
95	96	95	93	96	94	96	95	99	97	97	98	99	98	98	98	98	99	99	99	98	98	6.25	9.5	21	22	120	330	450
95	95	94	93	93	93	92	92	97	98	99	99	98	98	97	97	97	97	97	97	97	97	6.5	9	22	18	115	315	645
94	95	95	93	96	94	93	92	97	98	98	98	97	97	99	97	97	97	97	98	98	97	6	9.5	20	19	115	345	435
91	94	92	93	91	91	93	92	98	97	97	97	98	98	97	98	96	96	97	97	97	98	6.25	9.75	24	22	120	315	440
93	94	95	93	95	95	95	94	97	98	97	97	97	97	98	98	98	98	98	99	97	97	6.5	8.5	25	25	90	330	405